

Kidney type of DI

A plain-English guide for you and your family

WHAT IS HAPPENING

Your kidneys are like smart filters that clean your blood and make pee. Normally, a hormone called ADH (made by a tiny gland at the base of your brain) travels through your blood to your kidney and tells special channels in the kidney tubes to open up and pull water back into your body, so your pee gets stronger and you don't lose too much water. In nephrogenic diabetes insipidus, the kidney tubes cannot hear or respond to that ADH signal — either because certain medicines (like lithium, used for mood swings) or chemical imbalances (too much calcium or too little potassium in the blood) poison the listening posts on the kidney cells, or because a child is born with a broken gene (often on the X chromosome) that makes faulty water channels or broken ADH receivers. When the kidney ignores ADH, the tubes stay wide open and water keeps pouring out as very dilute, watery pee — sometimes 3 to 20 liters a day, which is like filling a big soda bottle every few hours. The body notices it is getting dry, so the brain's thirst center screams 'drink!', and the person feels an overwhelming need to gulp water all day and night. If they can drink enough, the blood stays mostly balanced, but if water is restricted — say, during sleep, illness, or a hospital test — the blood gets too salty (high sodium), which can confuse the brain, cause weakness, seizures, or even coma. This is not the same as 'sugar diabetes' (diabetes mellitus); the name 'diabetes insipidus' just means 'passing through tasteless urine' in old medical language, because the pee is so dilute it has almost no flavor, unlike the sweet pee in sugar diabetes. The key idea: the kidney's water-saving switch is broken, so the body loses water it should be keeping.

What you might feel

- You pee a huge amount every day — often 3 to 20 liters (like filling a big water jug, or even more than that)
- You feel thirsty all the time and drink water constantly
- You wake up many times at night to pee (nocturia)
- Your pee is very pale or almost clear
- Peeing and drinking keep happening even after you take desmopressin (DDAVP, a medicine that acts like ADH)

- Babies may be fussy, have fevers, vomit, or not gain weight well
- Your blood sodium goes high (hypernatremia — over 145 mEq/L) if you can't drink enough to keep up

Call the doctor right away if...

- Blood sodium above 150 mEq/L (very salty blood — can cause confusion, seizures, or coma)
- A baby who is limp, won't wake up, has sunken eyes or a sunken soft spot, or stops making tears (severe dehydration)
- Sudden confusion, muscle twitching, or seizures in anyone with known diabetes insipidus (sign the brain is being hurt by salt imbalance)
- Not making any pee for hours despite drinking (might mean the kidneys shut down from severe dehydration or another emergency)

What the care team will do

- Ensure the patient has unrestricted access to water or fluids at the bedside and throughout the care area at all times
- Monitor serum sodium level every 4 to 6 hours during acute management and watch for confusion or lethargy signaling high sodium (hypernatremia)
- Measure and record urine output every hour and compare to fluid intake to identify negative fluid balance early
- Check serum calcium and potassium levels daily and notify the provider immediately if calcium is above 10.5 mg/dL or potassium is below 3.5 mEq/L
- Hold lithium doses and notify the provider if lithium is on the medication list, then monitor for improvement in urine volume over 48 to 72 hours
- Administer prescribed thiazide diuretic (usually hydrochlorothiazide 25 to 50 mg daily) and monitor for reduced urine output within 3 to 5 days
- Teach the patient and family to follow a low-sodium diet (less than 2 grams of sodium daily) and provide a printed list of high-salt foods to avoid
- Weigh the patient at the same time daily and report weight loss greater than 2 pounds in 24 hours as a sign of dehydration

Medicines you may be given — and why

Thiazide diuretic (for example, hydrochlorothiazide)

This water pill blocks salt reabsorption early in the kidney tube (in the distal convoluted tubule). When less salt is pulled back into the blood, the body holds onto water earlier in the

tube to keep salt balance steady. That means less water reaches the collecting duct at the end, so less water is lost as urine—even though ADH (the hormone that normally tells the kidney to hold water) isn't working right. It is paradoxical: a diuretic that actually reduces urine volume in nephrogenic DI.

Amiloride (potassium-sparing diuretic)

Amiloride blocks sodium channels (ENaC) in the collecting duct, the same channels that lithium can clog up and damage. By blocking those channels, amiloride stops lithium from entering the cells and harming them. This protects the kidney cells that are supposed to respond to ADH. It also gently reduces urine volume by changing how the kidney handles salt and water. It is especially useful in lithium-induced nephrogenic DI.

Indomethacin (NSAID anti-inflammatory)

Indomethacin blocks an enzyme called cyclooxygenase (COX) that makes prostaglandins. Prostaglandins in the kidney normally oppose ADH—they make the kidney dump more water. By blocking prostaglandins, indomethacin allows whatever weak ADH signal is present to work a bit better. It also reduces blood flow to the kidney slightly, which cuts down the amount of water filtered in the first place. Together, these effects can reduce urine volume by 25–30%.

Stop lithium (if nephrogenic DI is lithium-induced)

Lithium enters kidney cells through sodium channels and interferes with the signaling cascade that ADH triggers inside the cell. Specifically, it blocks the insertion of aquaporin-2 water channels into the cell membrane, so even when ADH binds its receptor, the cell cannot open the gates to let water back into the blood. Stopping lithium removes the toxin and allows the kidney cells to start repairing. Recovery can take weeks to months, and some damage may be permanent if lithium was used for many years.

Correct high calcium (hypercalcemia) (high calcium) if present

High calcium in the blood directly interferes with the kidney's ability to respond to ADH. Calcium activates a receptor (calcium-sensing receptor, CaSR) on kidney cells that reduces the production and insertion of aquaporin-2 water channels. High calcium also triggers calcium deposits in the kidney tubules, which physically damage the cells. Lowering calcium—by treating the underlying cause (e.g., hyperparathyroidism, vitamin D toxicity, cancer)—removes this block. ADH signaling improves, and urine volume drops.

Correct low potassium (hypokalemia) (low potassium) if present

Low potassium impairs the kidney's ability to make and move aquaporin-2 water channels to the cell membrane in response to ADH. It also triggers changes in the cell that make it less sensitive to ADH signaling. Replacing potassium—by mouth (potassium chloride pills or

powder) or IV if severe—restores normal cell function. The kidney can then respond to ADH again, and urine volume drops.

Adequate water intake (cornerstone of all management)

Because the kidney cannot hold onto water, the body loses huge amounts of dilute urine every day (often 3–20 liters). The only way to prevent life-threatening dehydration is to drink enough water to match what is lost. This does NOT fix the kidney problem, but it keeps the person alive and prevents the blood from becoming dangerously concentrated (hypernatremia). Free access to water at all times is essential. Infants and people who cannot sense thirst or communicate need careful monitoring and scheduled water intake.

Low-sodium diet (essential adjunct to diuretic therapy)

A low-sodium diet (usually <2–3 grams of sodium per day, or <5–7 grams of salt per day) reduces the amount of sodium filtered by the kidney. When less sodium is delivered to the distal tubule, the kidney reabsorbs more water upstream (in the proximal tubule) to keep the body's salt balance steady. This reduces the volume of urine by 30–50% and makes thiazide and amiloride work much better. Without a low-sodium diet, diuretics often fail to reduce urine volume meaningfully.

Where this information comes from

- Endocrine Society Clinical Practice Guideline on Diabetes Insipidus
- UpToDate (Wolters Kluwer peer-reviewed synthesis)
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
- American Academy of Pediatrics (AAP) guidance on polyuria in children

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